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MOTION SICKNESS INCIDENCE AS A FUNCTION OF THE FREQUENCY AND ACCELERATION OF VERTICAL SINUSOIDAL MOTION

James F. O'Hanlon, et al

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13. ABSTRACT

Fourteen experimental conditions of vertical sinusoidal motion were defined by combinations of wave frequency and acceleration level in a partial factorial design. The frequency range investigated was from 5 cycles per minute (CPM, or .083 Hz) to 30 CPM (.500 Hz), and the average acceleration over each half-wave cycle ranged from about .03 to .40 g. Independent groups of 20 or more male Ss were exposed for 2 hours or until they began to vomit, whichever came first.

Motion sickness incidence (MSI), defined as the percentage of Ss experiencing vomiting, was greatest at a frequency of 10 CPM (.167 Hz). For all wave frequencies, MSI increased as a monotonic function of the acceleration level. A mathematical model was derived from the data, and the implications for underlying physiological mechanisms and for transportation vehicle design were discussed.

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TABLE OF CONTENTS

√

LIST OF TABLES AND FIGURES

MOTION SICKNESS INCIDENCE AS A FUNCTION OF THE FREQUENCY AND ACCELERATION OF VERTICAL SINUSOIDAL MOTION

INTRODUCTION

The incidence of motion sickness in man probably began when he first ventured upon the sea in ships. In the past century the signs and symptoms of motion sickness have been observed in other transportation vehicles: automobiles, aircraft, and spacecraft. Despite the various environments in which motion sickness may occur, a common characteristic of all the motions which induce motion sickness seems to be repetitive linear or angular acceleration of the head[1].

One component of the complex motions of ships, aircraft, and land vehicles is periodic vertical motion. This component of sea motion has long been considered as a primary factor in the etiology of motion sickness aboard conventional seacraft[2,3], but little is known about the characteristics of periodic vertical motion which induce the pathogenic effects. Practically all of the available data arise from a series of studies conducted under the direction of G. R. Wendt at Wesleyan University in the 1940's[4,5,6,7,8]. Although that research program was pioneering, it was limited in several important respects which are discussed later in this report. After a 25-year hiatus, that line of research was resumed in an attempt to identify the components of vertical periodic motion which induce motion sickness, and to show quantitatively how the incidence of motion sickness varies as a simultaneous function of those components.

1

METHOD

Subjects

The subjects (Ss) were 30 healthy, young (18-34 years) male college students who volunteered for pay ($10). There were, in addition, six volunteers who were rejected due to physical conditions revealed by their responses to a medical history questionnaire. In response to another questionnaire, none of the Ss indicated having been exposed to significant seacraft or aircraft motions within the preceding two-month period; i.e., all were apparently unacclimatized to motion.

Apparatus

The Ss were exposed, in pairs, to preselected motions in the ONR/HFR Motion Generator[9,10]. That device is capable of displacing a 2.44 m-square cabin over a 6.7 m vertical range. The cabin is mounted upon a carriage which rides over vertical tracks on a 9 m tower. The carriage is driven from beneath by a hydraulic piston. The piston's excursion is determined by a hydraulic positioning servomechanism under remote electronic control by apparatus contained in the adjacent experimenter's cabin. The frequency response of the entire system is across the range 0-1 Hz. The system is capable of producing maximum accelerations in the cabin of up to about 0.7 g.

The cabin's internal space was bisected into separate compartments by a floor-to-ceiling insulated partition. Access to each compartment was gained through a separate door and no communication was permitted between compartments. An aircraft-type seat with a lap belt and headrest was mounted in each compartment. Voice communication was achieved between the experimenter and each S via separate earphone/microphone systems mounted in headsets. Two closed-circuit TV systems allowed the experimenter to visually monitor the Ss. The compartments were maintained at a dry-bulb temperature of 21.2°C to 22.2°C by independent air conditioning systems.

Procedure

Originally, 280 Ss were randomly assigned in groups of 20 to participate in each of 14 experimental conditions. In three conditions additional Ss were randomly assigned in order to achieve greater statistical reliability of the ensuing data. Each condition was defined by a particular combination of wave frequency (f) and acceleration level. The measure of acceleration was the time integral of the absolute value of acceleration ($\bar{a}$) imparted in each half-wave cycle.*

The 14 experimental conditions, defined by combinations of f and $\bar{a}$ are given in Table 1, along with the corresponding displacement values and the number of Ss in each condition. For conditions employing a given f level, wave amplitude (i.e., ½ total displacement) was adjusted to yield the desired $\bar{a}$ levels in different conditions. As may be inferred from the table, more than 14 combinations of the given f and $\bar{a}$ levels would have been required to achieve a completely balanced factorial experimental design. However, those additional treatment conditions were both impossible to achieve with the present apparatus and were highly unrealistic with respect to vertical periodic motion encountered in conventional seacraft. As it was, the conditions that were used included vertical displacements between .30 m and 5.66 m, therefore bracketing the range of ocean wave heights in Sea States 1-6[11].

Subjects were instructed that their exposure would last for two hours, but that they would be removed sooner in case of emesis (vomiting). After instruction, the two Ss were seated within their respective compartments and their heads positioned against the headrests. No earth-fixed visual reference was

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*It has been a frequent practice to stipulate acceleration during sinusoidal motion in terms of the absolute value of peak acceleration (a_{max}); or alternatively, as the root-mean-square of acceleration in each half-wave cycle (a_{rms}). However, $\bar{a}$ seems to represent the physiologically effective stimulus better, and for that reason it was used here. The transformation of $\bar{a}$ into the other measures of acceleration is simple for sinusoidal motion (i.e., $\bar{a} = .637\, a_{max} = .901\, a_{rms}$).

available, and the interior cabin lights remained on. The *S*s were not prohibited from closing their eyes, but sleeping was not permitted; the *S*s were required to press one of five buttons every minute, upon presentation of a tone, to describe their approximate state of symptom development. The results of these data are not included in the present report.

The experimenter aurally and visually monitored the *S*s, using the apparatus described, to ensure their safety and to stop the motion generator if either *S* vomited. Upon emesis, the affected *S* was removed from his compartment and, after a delay of 30-60 seconds, the exposure was resumed for the other *S*.

The motion sickness incidence (MSI) in each condition was measured by the percentage of *S*s who experienced emesis at any time during the period of exposure.

TABLE 1

EXPERIMENTAL DESIGN WITH FULL-WAVE DISPLACEMENT IN METERS (TOP)
AND NUMBER OF SUBJECTS (BOTTOM) FOR EACH CONDITION

Acceleration $\bar{a}$ (g)

Frequency (CPM) (Hz)	.025	.05	.10	.20	.30	.40
5 .083	2.80 N=20	5.60 20				
10 .167	.70 20	1.40 20	2.80 20	5.60 20		
20 .333		.35 20	.70 26	1.40 27	2.10 33	
30 .500			.30 22	.61 20	.91 20	1.22 20

5

RESULTS AND DISCUSSION

Table 2 lists the MSI values recorded in each experimental condition. One aspect of those results was a monotonic increase of MSI, with $\bar{a}$, at every level of f. Attempts were made to fit a general equation to the data for describing MSI as a function of $\bar{a}$, or a logical transform of $\bar{a}$. It was determined that the equation for the integral of the normal distribution function (i.e., the normal ogive) accurately described the relationship between MSI and $\log \bar{a}$. The general expression was:

$$MSI = \int_{-\infty}^{\log \bar{a}} \frac{100}{\sigma\sqrt{2\pi}} \, e^{-[(x-\mu)^2/2\sigma^2]} \, dx$$

where π and e are defined as usual, x is a variable of integration in units of $\log \bar{a}$, and σ and μ are parameters with values determined empirically.

Parameter estimation was accomplished by fitting the general equation to the MSI values measured at every $\log \bar{a}$ level, for each level of f separately, according to the method of least-squares. The results obtained for the three higher levels of f are shown in Figure 1.*

The parameter, μ, corresponds to the $\log \bar{a}$ value associated with a 50% MSI value for a particular f level. Values of μ obtained at different f levels also indicate the relative effectiveness of $\log \bar{a}$ as a stimulus for motion sickness at those wave frequencies: lower μ values indicate that less acceleration is required to produce the same MSI.

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*Only two levels of acceleration were used in conjunction with the lowest f level (.083 Hz). These yielded only two data points too few for use in parameter estimation. It was is ed that any general relationship between MSI and $\log \bar{a}$ determined from the other data would hold for the lowest f level as well.

TABLE 2
MSI VALUES (PERCENT EMESIS) RECORDED IN EACH MOTION CONDITION

Acceleration $\bar{a}$ (g)

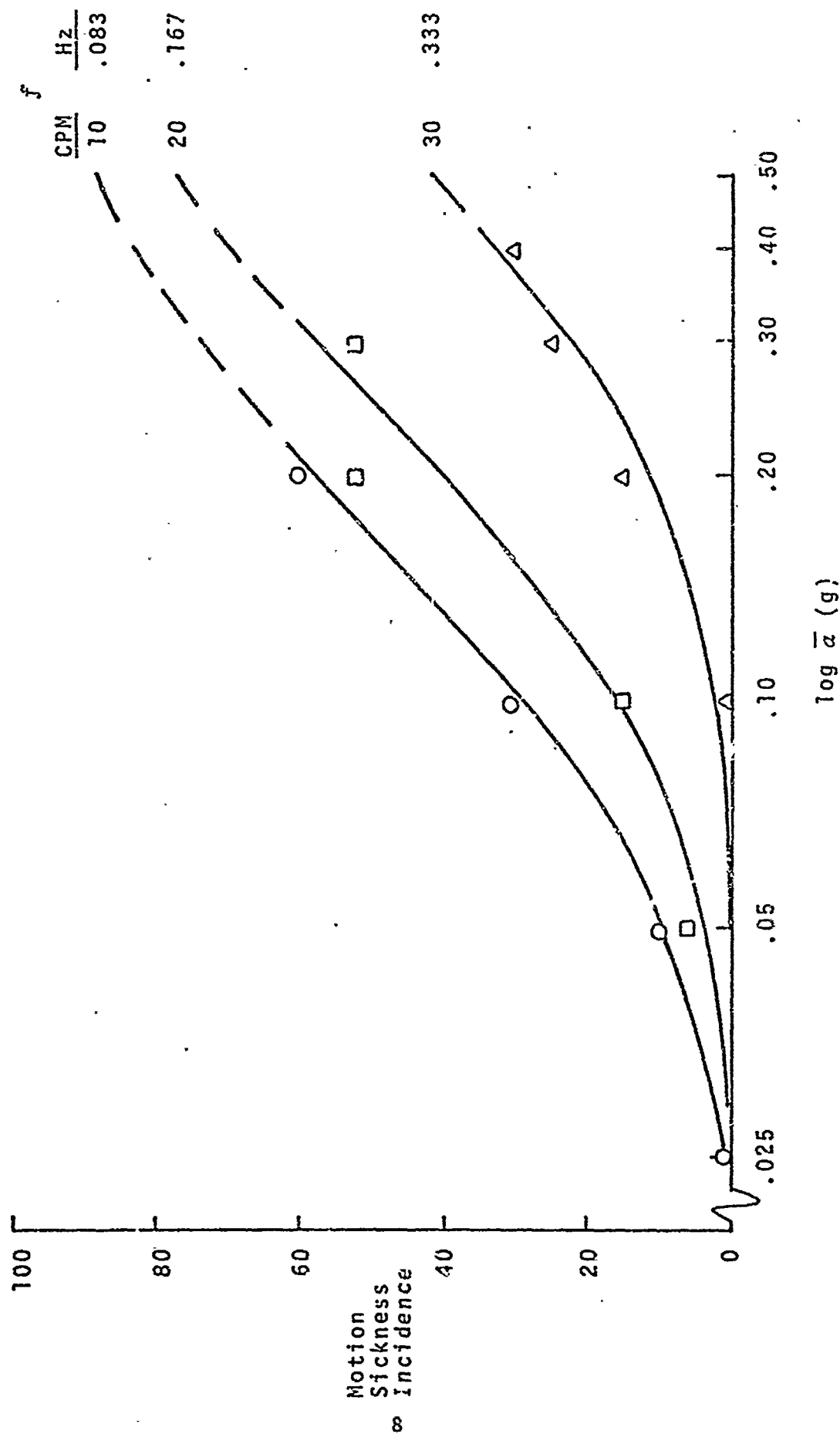| Frequency (CPM)	(Hz)	.025	.05	.10	.20	.30	.40
5	.083	0	5				
10	.167	0	10	30	60		
20	.333		5	15	52	52	
30	.500			0	15	25	30

Figure 1. Motion Sickness Incidence (within two hours) as a function of log average acceleration ($\bar{a}$) for each wave frequency (f).

The data indicated that µ increased with f between .167
and .500 Hz. Yet µ cannot be a monotonic function of f. Logi-
cally, µ must also increase as f declines from .167 Hz to zero.
The true relationship between µ and f may be complex, but an
initial approximation of that relationship was made by fitting
the quadratic equation to the data according to the method of
least-squares, so that:

$$\mu = .659 + 3.840 \log f + 2.467 (\log f)^2$$

where f is in Hz and µ is a log $\overline{a}$ scale with $\overline{a}$ in units of g.

By substituting the observed value of .40 log $\overline{a}$ for σ, and
by substituting the above expression for µ in the first equation,
a model was derived for expressing MSI as a function of $\overline{a}$ and f.
A graphic representation of that model is shown in Figure 2,
along with the actual data used in the derivation. The 14 data
points are the same as previously presented in Table 2. The
deviation of each of the actual data points from the corresponding
points on the derived surface is shown in Figure 2 by the dashed
interconnecting lines. Each dash and each space represents a 1%
difference between the actual and derived points. As shown, the
curved surface fits the actual data in a satisfactory manner.
The MSI observed in only one group (.20g $\overline{a}$ and .333 Hz f) deviated
from the corresponding point on the derived surface by more than
6%. Moreover, the root-mean-square of deviations of the actual
data points from the respective derived points was only 3.96%,
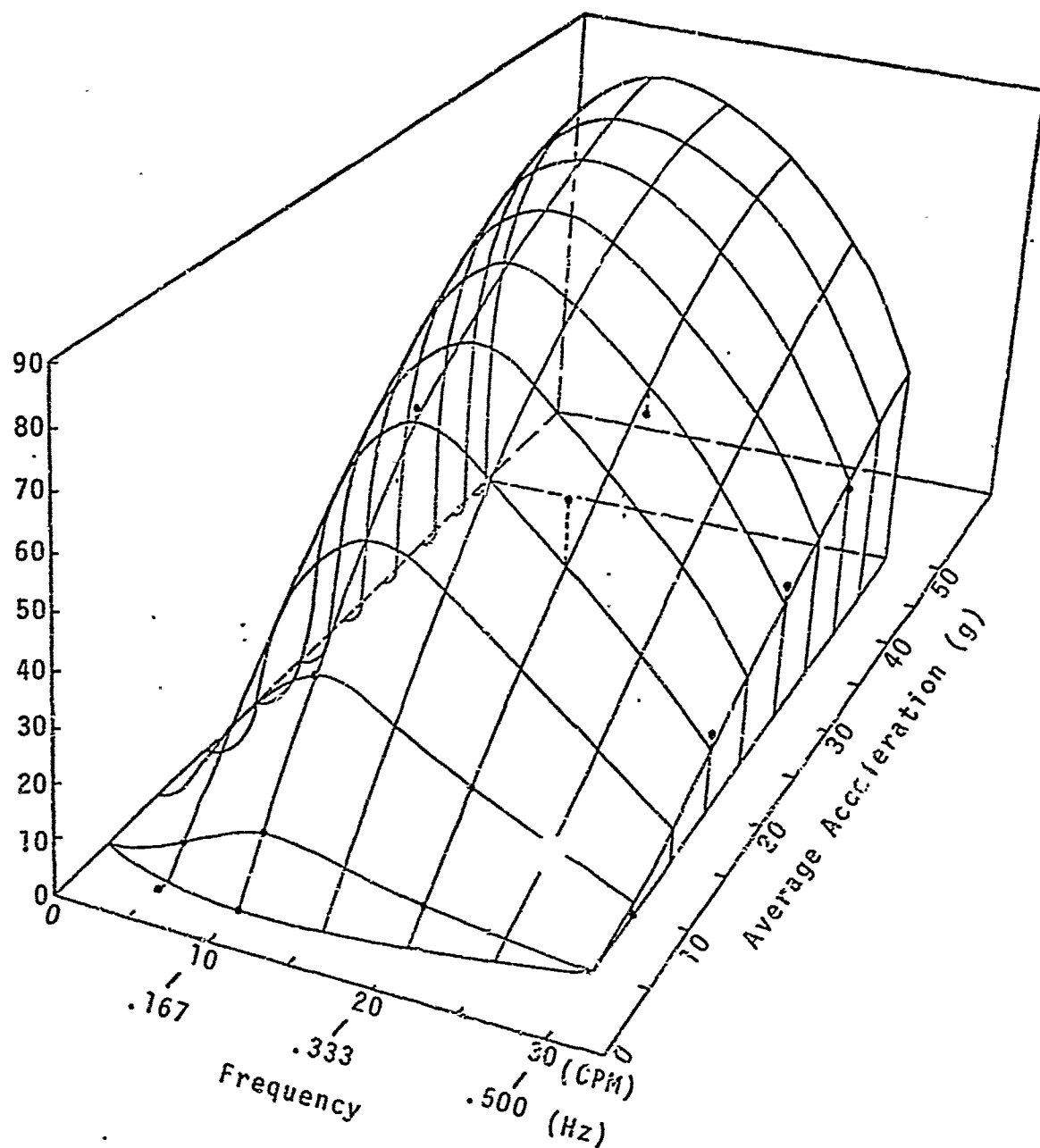indicating again that the model closely fits the data.

Figure 2. Empirically derived relationship of MSI (percent emesis within two hours) to wave frequency and average acceleration imparted during each half-wave cycle for vertical sinusoidal motion.

10

GENERAL DISCUSSION AND CONCLUSIONS

In its present rudimentary stage, the model has limited
practical utility. It ignores the obviously important factors
of exposure period and acclimatization to motion. Our guess
is that progressive acclimatization would systematically reduce
the overall height of the MSI surface without changing its basic
shape in either the f or $\bar{a}$ dimensions.

In spite of its limitations, the existing model may have
potential application for the design of desirable ride character-
istics in future land, sea, and aircraft. Our data indicate
that even moderate accelerations at frequencies near .2 Hz should
be avoided as these produce the highest incidence of motion sick-
ness. Humans can apparently tolerate higher accelerations at
higher frequencies (e.g., .5-1.0 Hz) without experiencing the
same tendency toward motion sickness. An engineering strategy
to "smooth out" a ride should be considered cautiously if high-
frequency motion (over .5 Hz) is to be reduced at the expense of
increasing the energy in the lower frequency bands associated
with motion sickness. One should avoid applying such a strategy
when there is a danger of producing a ride which leads to an un-
necessarily high MSI.*

In historical perspective, the work begun by Wendt and his
associates was extended in this study. The earlier work has been
criticized for providing results of limited generality due to
procedural inadequacies[13,14]. Cited in particular were the use
of very short (i.e., 20-minute) exposure periods and artificial
wave-forms (i.e., the motions provided alternating periods of
constant velocity--constant acceleration). Nonetheless, with

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*Kennedy *et al.*[12] have previously offered essentially the same
advice. They postulated the existence of a relatively benign
frequency range between frequencies which produce motion sick-
ness (i.e., < 0.5 Hz), those which approximate the resonance
frequency of the human body (i.e., 4.0-8.0 Hz). In their view,
a goal of design engineers should be to ensure that as much as
possible of the total energy imparted to the occupants of vehi-
cles is within the benign frequency range rather than within the
immediately higher or lower bands.

11

improved procedures, we found essentially the same curvilinear relationship between wave frequency and MSI, as did the early workers. But, by testing lower frequencies while holding acceleration constant, our data show the peak of the curve (i.e., the most pathogenic frequency) to be about 10 CPM as opposed to their estimate of 22 CPM[6]. It seems safe to conclude that wave frequency is a critical factor for determining the response of the physiological mechanism responsible for motion sickness in vertical periodic motion, and that its maximum responsitivity seems to be in the frequency region around .2 Hz.

The Wesleyan group never systematically studied the effects of wave acceleration, and thus had no opportunity to observe the striking relationship between average wave acceleration and MSI. Regardless of wave frequency, MSI increased with $\bar{a}$. When the MSI was plotted as a function of log $\bar{a}$ for each f level separately, an apparently general relationship emerged. That relationship bears an intriguing resemblance to the psychophysical relationship between a behavioral response indicating the sensation of a near-threshold stimulus and the physical intensity of that stimulus. The psychophysical relationship is described by the classic phi-log gamma function, which has been frequently observed while plotting the cumulative probability of correct discriminations (phi) against the log stimulus intensity (gamma)[15]. The phi-log gamma function is a normal ogive which is thought to arise because the underlying probability distribution of sensory thresholds is normal, both within and between individuals, with a mean of μ and a standard deviation of σ--again, on a log scale of stimulus intensity.

By way of analogy, emesis may be considered as a behavioral response indicating that a motion sickness threshold has been exceeded within an individual. The demonstrated ogival relationship between MSI and log $\bar{a}$ could then be viewed as a phi-log gamma function, implying an underlying normal distribution of emesis thresholds in the subject population for a given wave frequency

and exposure period. The model further implies that the mean
of that distribution is frequency dependent, and that the variance
of population thresholds is constant with resp   to frequency--
on a log $\bar{a}$ scale.

There are several theoretical implications of this reasoning
for motion sickness in vertical periodic motion. I implies that
the operating characteristics of the physiological mechanism re-
sponsible for emesis are similar to those of better understood
sensory processes. Furthermore, it implies that the emesis
threshold in every normal individual will eventually be exceeded
as log $\bar{a}$ increases, for any frequency within the motion sickness
region (unless, of course, other pathologic effects of high accel-
erations intervene).

In conclusion, a preliminary model has been provided which
simultaneously relates MSI to the frequency and acceleration
parameters of vertical periodic motions. Though limited, the
model can be used immediately for evaluating the relative patho-
genicity of commonly encountered combinations of wave frequency
and acceleration. It may also provide new insight regarding the
operating characteristics of basic physiological mechanisms under-
lying motion sickness.

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